ABSTRACT OF THE DISCLOSURE

Anterior ischemic optic neuropathy (AION) is one of a family of ischemic diseases affecting the optic nerve. A blockage of vessels supplying the intra-retinal portion of the optic nerve results in loss of axon transport stasis, retinal ganglion cell (RGC)--specific dysfunction, and RGC death. AION research has been limited by the lack of an appropriate, easily replicable, rapidly inducible, low-cost model for this disease. We have developed such a model. Animals were handled and utilized in accordance with ARVO guidelines. Using a custom designed fundus contact lens, an intravenous injection of photosensitizing agent was administered to anesthetized 100g male Sprague-Dawley rats. A laser was used to directly activate dye within the small vessels perfusing the optic nerve. This treatment was adjusted to selectively spare the larger caliber vessels perfusing the inner retina. Gross, histologic, molecular, and electrophysiological techniques are used to analyze changes induced by this method. The acutely treated rodent optic nerve grossly has the appearance of human AION, with pale edema. Electrophysiological, a decease in amplitude of the visual evoked potential is noted. Histologically, alterations in axonal transport are seen. Reverse-transcriptase based polymerase chain reaction (rt-PCR) indicates that there are both early RGC-specific, and later retinal gene expression changes in the treated animals. This new method accurately

replicates many cellular and molecular level in a low-cost animal model may greatly accelerate our understanding of the pathological long and short term processes involved in AION, and increase our ability to develop more effective treatments for this disease.